Case Reports

Mad-Honey Sexual Activity and Acute Inferior Myocardial Infarctions in a Married Couple

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Mad-honey poisoning can occur after the eating of honey that contains grayanotoxin. Mad honey is intentionally produced from the nectar of Rhododendron ponticum*, which grows in Japan, Nepal, Brazil, parts of North America and Europe, and the eastern Black Sea region of Turkey. Low doses of grayanotoxin can cause dizziness, hypotension, and bradycardia, and high doses can cause impaired consciousness, syncope, atrioventricular block, and asystole due to vagal stimulation. Reports of acute coronary syndrome are very rare. Herein, we present the case of a 50-year-old husband and 42-year-old wife who, to improve sexual performance, intentionally ate honey from the Black Sea area of Turkey for 1 week. Within 3 hours of consuming increased amounts of the honey, they presented at our emergency department with acute inferior myocardial infarctions. Coronary angiography revealed normal coronary arteries in both patients. Supportive treatment with atropine rapidly resolved the clinical symptoms and electrocardiographic irregularities. Grayanotoxin-containing rhododendron pollen was detected in the honey.*

In patients from geographic regions where mad honey can be obtained, mad-honey poisoning should be considered in the differential diagnosis of chest pain, particularly in the presence of unexplained bradyarrhythmia and hypotension. Sexual performance is a chief reason for the purchase of mad honey and self-treatment with it by persons of our patients' ages. ***(Tex Heart Inst J 2011;38(5):577-80)***

Key words: *Atropine/therapeutic use; biological therapy/adverse effects; foodborne diseases/complications; heart rate/drug effects; honey/adverse effects/poisoning; libido/drug effects; myocardial infarction/chemically induced/diagnosis/etiology; pollen/poisoning; sexual behavior/drug effects; Turkey*

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Mad-honey poisoning can occur after eating honey that contains grayanotoxin. Grayanotoxin-containing honey is intentionally produced from the nectar of *Rhododendron ponticum*, which grows in Japan, Nepal, Brazil, parts of North America and Europe, and the eastern Black Sea region of Turkey.[1] Grayanotoxin has a toxic cellular effect on the sodium channel and leads to activation in the vagus nerve by increasing sodium-channel permeability.[2,3] Clinically, grayanotoxin in low doses has caused dizziness, hypotension, and bradycardia; high doses have caused impaired consciousness, syncope, atrioventricular block, and asystole due to vagal stimulation.[4-9] Identifying the cause of these symptoms relies upon taking a thorough medical history from the patient, and a conclusive diagnosis can be reached after analyzing the pollen in the honey.[10] Herein, we report the case of a married Turkish couple who presented emergently with acute coronary syndromes after intentionally consuming honey for reasons of sexual performance.

Case Report

In September 2008, a 50-year-old husband and his 42-year-old wife presented at our emergency department with confusion and chest pain. Their medical histories could not immediately be taken. The man's blood pressure was 70/40 mmHg and his heart rate was 35 beats/min; the woman's blood pressure was 85/55 mmHg and her heart rate was 45 beats/min. Electrocardiograms (ECGs) showed complete atrioventricular block, a ventricular rate of 42 beats/min, and ST-segment elevation in the inferior leads in the man (Fig. 1); and atrioventricular nodal rhythm, a ventricular rate of 42 beats/min, and ST-segment elevation in the inferior leads in the woman (Fig. 2). Neither ECG revealed right ventricular infarction. After both patients were given 1 mg of atropine, their blood pressures and heart rates recovered dramatically, and their chest pain and ST-segment elevations resolved within 5 minutes. In the catheterization laboratory, coronary angiography showed normal coronary arteries in both patients

(Figs. 3 and 4). Both were then taken to the coronary intensive care unit, where ECGs 30 minutes after admission showed complete ST-segment resolution in the inferior leads. By this time, the patients were clinically and hemodynamically stable. Levels of cardiac troponin I were 18.7 ng/mL in the man and 13.4 ng/mL in the woman 6 hours after the onset of their chest pain. The couple revealed that they had eaten mad honey 3 hours before admission to the hospital. During their weeklong consumption of the honey for reasons of sexual performance, mild headaches and dizziness had occurred, but they had not cared. They had increased their intake of honey from 1 teaspoon each to 1 tablespoon each on the morning of their hospitalization.

Analysis of the honey revealed grayanotoxin-containing rhododendron pollen. The patients were discharged on the 5th day of hospitalization, and no medications were prescribed. One month later, both were asymptomatic, and results of ECGs and echocardiography were normal.

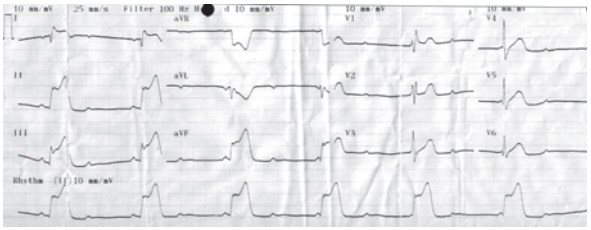

Fig. 1 *Male patient. Electrocardiogram upon hospital admission shows complete atrioventricular block and ST-segment elevation in the inferior leads.*

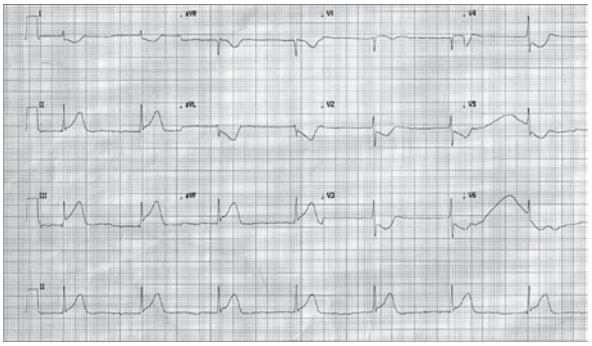

Fig. 2 *Female patient. Electrocardiogram upon hospital admission shows atrioventricular nodal rhythm and ST-segment elevation in the inferior leads.*

Discussion

Mad-honey poisoning is caused by the ingestion of grayanotoxin. Honey with grayanotoxin is intentionally produced from the nectar of *Rhododendron ponticum*, which grows extensively in the mountains of the eastern Black Sea region of Turkey.[1] The toxic cellular effect of grayanotoxin is on the sodium channel.[2] Maejima and colleagues[3] stated that grayanotoxin has 3 actions on the voltage-dependent sodium channel. First, grayanotoxin binds to the voltage-dependent sodium channel in its open state. Second, the modified sodium channels are unable to inactivate. Third, the activation potential of the modified sodium channel is shifted in the direction of hyperpolarization. Thus, grayanotoxin creates increased sodium channel permeability and activates the vagus nerve, causing dizziness, hypotension, and bradycardia in low doses, and impaired consciousness, syncope, atrioventricular block, and asystole in high doses due to vagal stimulation.[4-9]

A clinical evaluation that includes close questioning of the patient is typically sufficient to establish the diagnosis of mad-honey poisoning; but in difficult cases,

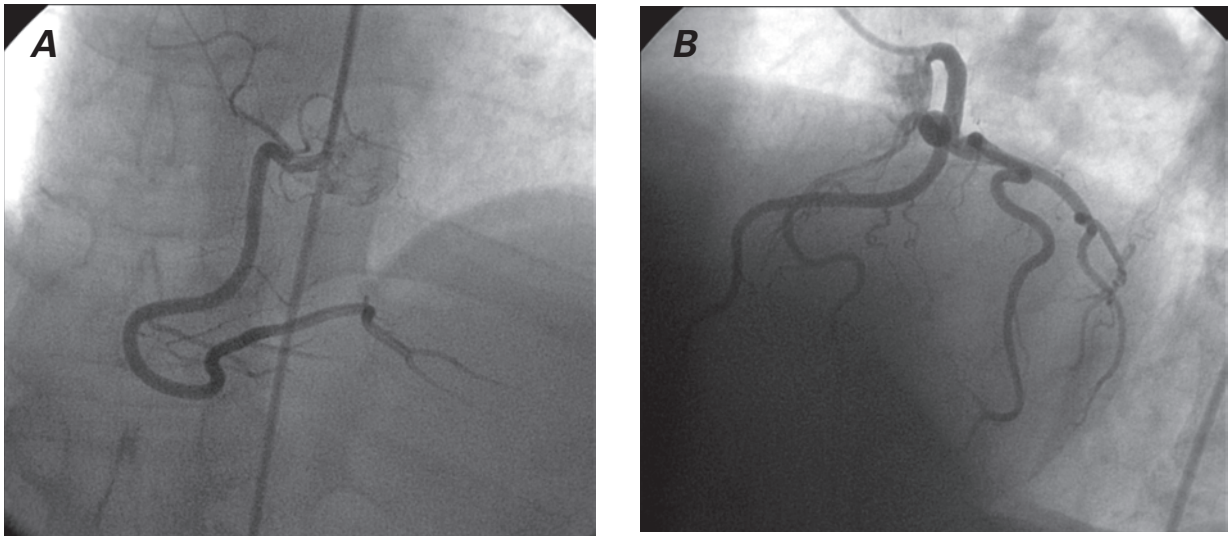

Fig. 3 *Male patient. Coronary angiography shows no evidence of flow-limiting lesions in **A**) the right coronary artery or **B**) the left coronary arteries.*

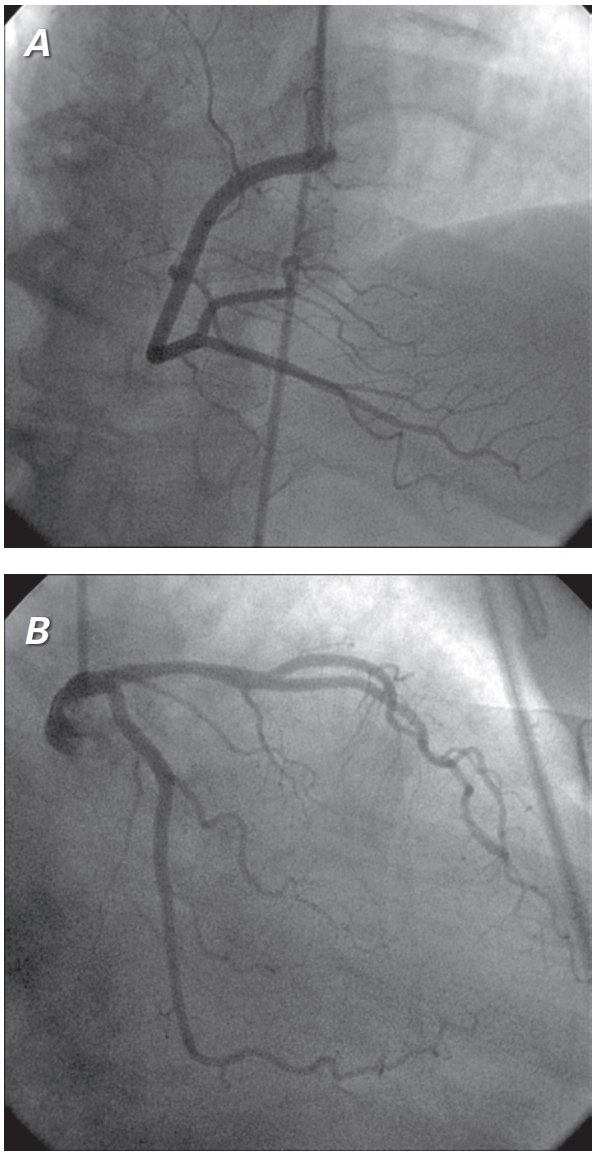

Fig. 4 *Female patient. Coronary angiography shows no evidence of flow-limiting lesions in* ***A****) the right coronary artery or* ***B****) the left coronary arteries.*

or when a dietary and travel history cannot be taken, pollen analysis can be definitive.[10] In our patients, mad-honey poisoning probably led to myocardial infarctions (MIs) by causing excessive bradycardia and hypotension. Acute inferior MIs occurred within 3 hours after the patients ingested increased amounts of the honey; after atropine was given, their blood pressures and heart rates returned to normal, and their angina and ST-segment elevations resolved. Upon analysis, the rhododendron pollen in the honey was determined to be the precipitant of the patients' acute MIs.

To our knowledge, no fatal cases of mad-honey poisoning have been reported since ancient Roman times. Typically, the effects of inadvertent poisoning last no longer than 24 hours, and supportive care is sufficient as treatment. Severely depressed blood pressure usually responds to atropine and saline infusion therapy; vasopressor therapy is rarely required.[11] In the mechanisms of acute coronary syndrome from the eating of mad honey, there has been no evidence of diminished endothelial completeness or of traditional thrombotic processes. Therefore, our patients did not require long-term antiplatelet therapy.

In persons with normal coronary arteries, prolonged vagal tonus may lead to transient ST-segment elevation because of hypotension and bradycardia.[12,13] Arita and colleagues[12] reported transient ST elevation in the inferior ECG leads due to a Bezold-Jarisch-like vasovagal response that was mediated by the mechanical effects of puncture on the vagal network during the Brockenbrough procedure for radiofrequency catheter ablation. In our patients, the main mechanism of the transient ST elevation probably depended upon a prolonged vagal tonus that was induced by the grayanotoxin in the honey. Their rapid recovery after atropine administration also supports this reasoning.

To our knowledge, acute coronary syndrome from mad-honey poisoning has been reported only twice. Inferior ST-segment-elevation MI (STEMI) was diagnosed in 1 case,[14] and non-STEMI with ST depression in the anterior ECG leads was diagnosed in the other.[15] Our patients' cases support the scant published evidence that mad-honey poisoning can lead to acute coronary syndromes. The earlier reports suggest that mad-honey poisoning should be kept in mind as a differential diagnosis in the presence of STEMI and the absence of coronary lesions.

In a survey of Turkish beekeepers who produced and sold mad honey as an alternative medicine,[16] men aged 41 to 60 years purchased mad honey chiefly for self-treatment of sexual dysfunction. This was the second most common reason for purchase by women of that age. Our married patients in this report fit that demographic category.

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